Benign Paroxysmal Vertigo in Childhood

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A B S T R A C T

The aim of the paper is to describe the clinical picture of benign paroxysmal vertigo (BPV) in childhood. BPV in childhood often goes unrecognized in spite of the fact that vertigo and balance disorders are not uncommon in children. Four cases are described with clinical examination findings between the attacks.

***Key words**: paroxysmal vertigo in children*

Introduction

Benign paroxysmal vertigo (BPV) was first described by Basser in 1964[1]. The pathogenesis of BPV is still unknown. The onset is usually between three or four years of age, but may occur even later at the age of seven or eight. BPV attacks always occur spontaneously in healthy children with no signs of previous illnesses. The attacks cease spontaneously after several months or more frequently several years, without residual disability[2,6,10]. The main symptom is vertigo. Consciousness is not impaired during BPV attacks. Tinnitus, deafness and headaches are not present. Nystagmus is seen during the attacks in some cases but not between the attacks[1,14].

The attack may occur at any moment when children are standing, running, sitting or lying. Duration of the attacks is very brief, lasting less than a minute and rarely lasting more than a few minutes. During severe attacks children become pallid, frightened, very weak, incapable to move, and have intensive perspiration. Some of them have episodes of head-tilt[15]. During less severe attacks children run to their parents and strongly hold onto their clothing until the attack ceases. A short time after the attack, children recover completely, usually within 1 to 3 minutes. The attacks recur at varying intervals in children. They may occur every day, every week, after 6 to 8 weeks, or after 6 to 12 months. Children are well between the attacks. The attacks cease spontaneously after a few months, usually within a few years, without any residual disability.

No abnormalities are found in otological, neurological or radiological examinations. The abnormal finding is demonstrated by the caloric tests which show disorders of vestibular function in the form of complete unilateral or bilateral canal paresis. These disorders cease after the attacks[10,13]. EEG and NMR of the brain are normal.

At present, the theory that BPV might be a migraine equivalent or a migraine precursor is widely accepted[3,8,9,14]. A large number of follow-up studies show that most of the children suffering from BPV develop some features typical of migraine during adulthood[2,3]. Other studies show that most of the children have also a positive family history of migraine[10,19].

Case Reports

This study included 2 girls and 2 boys. The children were examined on an individual basis over a period of 7 years.

Case 1 (I. B.)

The boy had his first attack at the age of 4. Before that attack he was healthy and was feeling well. The attack occurred suddenly, without warning and lasted less than a minute. Before the attack the boy was playing in the garden. Suddenly he became frightened and pale. He ran to his mother, crying and clinging tightly to his mother's clothes. During the attack the boy did not lose

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consciousness. The main symptom was vertigo. There was no vomiting, tinnitus, deafness, nystagmus, or headaches during, after or between the attacks. Sweating was present during some attacks.

After the attack the child recovered quickly within 2 to 3 minutes and continued to do what it was doing previously. Between the attacks the child was feeling well. The attacks recurred every 6 months for the next 7 years and at the age of 11, they ceased spontaneously.

There was no history of otitis media. The medical examinations did not show any signs of otological, neurological and radiological abnormality.

The caloric test showed bilateral canal paresis. The electroencephalogram (EEG) and Nuclear Magnetic Resonance (NMR) of the brain were normal.

There was no personal or family history of epilepsy.

Case 2 (M. K.)

This girl had her first attack when she was three and a half years old. She was previously well and healthy. All her attacks were similar. They occurred without warning and lasted less than a minute. During the attack she was not able to move or do anything. The main symptom was vertigo. Consciousness was not impaired. There was no nystagmus, vomiting, headache, tinnitus, or deafness during, after or between the attacks. Sweating has been noted during some attacks. The complete recovery after the attacks was very quick, within 1 to 3 minutes. Between the attacks the girl was feeling well. The attacks occurred once a year and continued for the next 6 years and ceased spontaneously when she was nine and a half years old.

The medical examinations did not show any signs of otological, neurological and radiological abnormality. The caloric test showed normal findings on the right and canal paresis on the left side.

There was no personal or family history of epilepsy. The child's mother suffers from migraine attacks.

Case 3 (M. B.)

This girl had her first attack when she was nineteen months old. The attacks recurred repeatedly every two to six months and lasted between one and a half to two minutes. They spontaneously ceased at the age of 7. During the attacks, the girl could not stand up without support. She was pale, frightened and very weak. Consciousness was not impaired. Nystagmus and sweating were observed during 3 attacks. There was no vomiting, tinnitus or headaches. The complete recovery was within in 1 to 2 minutes after the attacks.

No abnormalities were found in the otological, neurological and radiological examinations between the attacks. There were no signs of tinnitus or nystagmus between the attacks. The caloric test showed canal paresis on the left side.

There was no personal or family history of epilepsy.

Case 4 (I. K.)

This boy had his first attack when he was seven and a half years old. He was completely healthy before the attack. On his way to school he suddenly sat on the ground because of vertigo attack. During the attack he was pale, frightened and sweated profusely.

The complete recovery was quick, in less than 3 minutes, and he was able to walk to school. The attacks occurred repeatedly every 4 to 6 weeks for the next 7 years, and they ceased spontaneously at the age of 14. All of the attacks were similar. They lasted less than a minute. There was no tinnitus, deafness, headaches, vomiting, or nystagmus during or after the attack. The main symptom was vertigo.

The boy was feeling well between the attacks, without otological, neurological and radiological examinations abnormalities. The caloric test showed canal paresis on the left side. There was no history of otitis media and no personal history of epilepsy. Two of the boy's aunts suffered from regular migraine attacks.

TABLE 1
BPV ATTACKS CHRONOLOGY

	First attack	Child's clinical status before attacks	Onset of attacks	Duration of attacks	Frequency of attacks	Child's clinical status between attacks	Attacks ceased
Case 1	4 years	Healthy	Without warning	Less than 1 minute	Every 6 months for the next 7 years	Without otological and neurological symptoms of diseases	Spontaneously
Case 2	3 and a half years	Healthy	Without warning	Less than 1 minute	Every 11–12 months for the next 6 years	Without otological and neurological symptoms of diseases	Spontaneously
Case 3	1 and a half year	Healthy	Without warning	Between and 1.5 and 2 minutes	Every 2–6 months for the next 5 years	Without otological and neurological symptoms of diseases	Spontaneously
Case 4	7 and a half years	Healthy	Without warning	Less than 1 minute	Every 4–6 weeks for the next 7 years	Without otological and neurological symptoms of diseases	Spontaneously

TABLE 2
MAIN CHARACTERISTICS OF BPV ATTACKS

	Main symptom	Conscious during attacks	Headache	Skin texture during attacks	Behaviour during/after attacks	Recovery after attacks
Case 1	Vertigo	Not impaired	None	Pale, with sweating	Frightened, very weak	Complete, very quick, within 2–3 minutes
Case 2	Vertigo	Not impaired	None	Pale, with sweating	Frightened, very weak,	Complete, very quick, within 1–3 minutes
Case 3	Vertigo	Not impaired	None	Pale, with sweating	Frightened, very weak	Complete, very quick, within 1–2 minutes
Case 4	Vertigo	Not impaired	None	Pale, with sweating	Frightened, very weak	Complete, very quick, within 2–3 minutes

TABLE 3
OTOLOGICAL EXAMINATIONS FINDINGS IN BPV PATIENTS

	Tympanic membrane	Tinnitus	Hearing	Audiogram	Nystagmus during/ between attacks	Caloric test
Case 1	Normal	None	Normal	Normal	None	Bilateral canal paresis
Case 2	Normal	None	Normal	Normal	None	Canal paresis on the left
Case 3	Normal	None	Normal	Normal	Yes/none	Canal paresis on the left
Case 4	Normal	None	Normal	Normal	None	Canal paresis on the left

TABLE 4
NEUROLOGICAL AND RADIOLOGICAL EXAMINATIONS FINDINGS IN BPV PATIENTS

	EEG	NMR of the brain
Case 1	Normal	Normal
Case 2	Normal	Normal
Case 3	Normal	Normal
Case 4	Normal	Normal

Discussion

The diagnosis of childhood BPV can be established on the basis of the typical anamnesis and absence of hearing disorders, absence of nystagmus between the attacks, absence of neurological disorders and normal NMR findings. Horizontal semicircular canal paresis or paralysis can be found in some children with BPV; in some the excitability of the horizontal semicircular canal remains normal[12,18].

We did not use the Vestibular Evoked Myogenic Potential (VEMP) during the diagnostic procedure in our patients. The dysfunction of the VEMP could be the sign of a saccular dysfunction, or a dysfunction of the reflex arc beginning with the saccule and going through the lower brainstem while reaching the sternocleidomastoid muscle. Some authors described the dysfunction of the VEMP in BPV[13,15,19], but the explanation of this phenomenon does not exist.

Recently some theories have been developed about ischemia of vestibular pathways as the factor triggering the BPV attack[7,16,19]. Cerebral vasospasms, as they occur in migraine could reduce labyrinthine artery perfusion and cause ischemia of the inner ear[4,11,17].

This theory is supported by high stimulus rate Brainstem Evoked Potentials (BEP) and the Transcranial Doppler (TCD) measurements. High stimulus rate BEPs are more sensitive to detect synaptic dysfunction than the usually used BEPs[4]. The TCD detects changes in cerebral blood flow velocity[5]. According to Zhang et al., high stimulus rate BEP as well as the TCD showed abnormal results in nearly half of the children suffering from BPV[19].

Conclusion

BPV in childhood goes often unrecognized. This problem should receive more attention because vertigo in children is more common than previously thought. The theory that BPV might be a migraine equivalent or a migraine precursor is widely accepted. In the pathogenesis of BPV, ischemia of the inner ear due to vasospasm may be involved as a triggering factor.

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BENIGNA PAROKSIZMALNA VRTOGLAVICA (BPV) U DJECE

SAŽETAK

Cilj ove studije bio je opisati Benignu paroksizmalnu vrtoglavicu (BPV) u djece, koja u većini slučajeva ostaje neprepoznata i zanemaruje se. U ovom članku prikazana je klinička slika BPV napada u četvero (4) djece. Mehanizam nastanka napada BPV još uvijek je nepoznat. Napadi BPV pojavljuju se najčešće u prve četiri godine života, rjeđe do osme godine života, u zdrave djece, iznenada, bez znakova upozorenja i traju od par sekundi do najduže 5 minuta. Napadi se mogu pojaviti u svakom momentu, dok dijete stoji, sjedi, leži, šeće, igra se itd. Dijete odmah prekine aktivnost u kojoj se nalazi, zastane ili sjedne, ne sruši se, jako se uplaši, problijedi i oznoji se. Vodeći simptom je uvijek vrtoglavica. Svijest je potpuno očuvana. Tijekom napada dijete dobro čuje, nema šum u ušima, nema glavobolje, a nistagmus se može i ne mora pojaviti. Mučnina i povraćanje nisu karakteristični za BPV. Nakon napada dijete se vrlo brzo i u potpunosti oporavi i nastavi raditi što je dotad radilo, kao da se nije ništa dogodilo. Ako je za vrijeme napada vrtoglavica slabijeg intenziteta, uplašeno dijete nesigurnim koracima potrči osobi koja ga čuva i čvrsto se uhvati za njenu odjeću. Napadi se ponavljaju, ali u svakog djeteta u različitom vremenskim intervalima i spontano nestaju nakon par mjeseci ili par godina. Dijete se između napada osjeća dobro i ne pokazuje znakove otološke i neurološke bolesti, što potvrđuju uredni nalazi otoskopskih pregleda, audiometrije, EEG-a i NMR mozga. Iznimka su kalorijski testovi koji pokazuju jednostranu ili obostranu parezu kanala. Nekoliko mjeseci nakon spontanog potpunog prestanka napada, ponovljeni kalorijski testovi pokazali su obostrano uredne nalaze u sva 4 ispitanika.